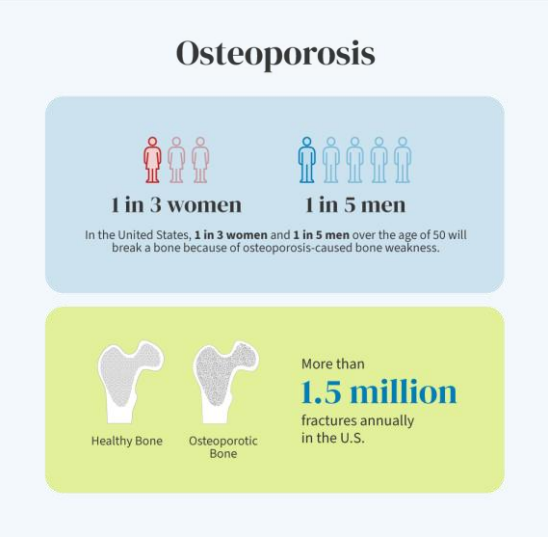
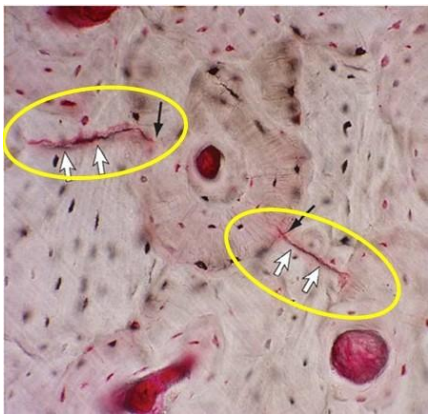
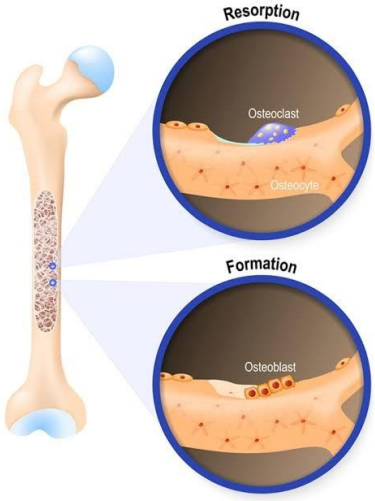
I Have Osteoporosis - Now What?



Osteoporosis and osteopenia are common after the age of 50 years, ***affecting 54%, the majority***, of that population. The primary concern with osteoporosis ***is fragility fracture*** which is a fracture that occurs spontaneously or from a more minor stress. It is that concern that indicates the need for treatment as the fractures result in significant disability and ongoing pain.

First, a little discussion about what causes bone loss with age as that helps to understand treatment choices. Osteoporosis is caused by a progressive imbalance in the bone remodeling cycle. Bone develops ***wear related microcracks*** which need to be repaired. The circled area in the diagram shows these microcracks. ***Remodeling*** replaces these cracked areas with ***new bone***.

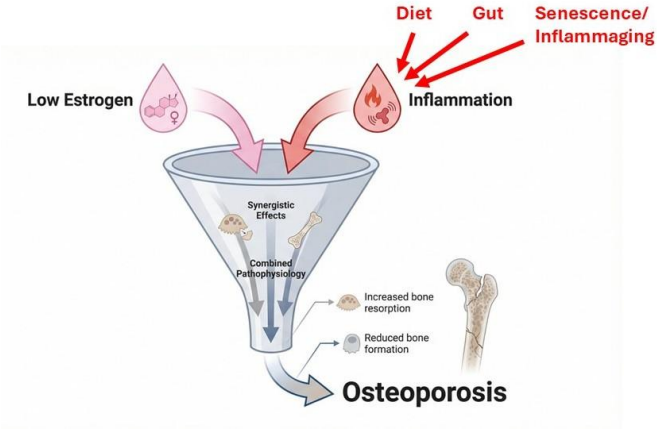




Each year 10% of the skeleton must be remodeled with the ***entire skeleton*** being replaced each decade. During this remodeling, ***osteoclasts*** first ***remove*** the bone in the area of wear. They are followed by ***osteoblasts filling it back*** in with new bone.

These cells tend to be ***in balance*** in their activity up to about age 35 replacing as much bone as is removed each cycle. Between age 35 and menopause, the osteoclasts begin to ***remove a little more*** bone each remodeling and the osteoblasts ***replace a little less***. The net effect is ***a loss of 1-2% of bone mineral density per year***. This imbalance in cell activity ***accelerates at menopause to a loss of 3-5% per year.***

An important question is what causes this imbalance in cell activity? Research suggests that it is related to 3 factors; ***declining estrogen***, ***genetics*** and ***inflammation***. ***Estrogen maintains the balance*** in osteoclast and osteoblast activity. As it begins to decline at about age 35, it allows the osteoclasts to get more active and the osteoblasts to get less active ***resulting in bone loss each remodeling cycle***.



Genetics is the least understood and modifiable cause. It is the reason why different people have different rates of bone loss and susceptibility to osteoporosis. It also can be the reason some people are less responsive to the different available treatments. The third factor, inflammation, is the ***most modifiable cause***. Inflammation increases the activity of osteoclasts which increases the bone removal each remodeling. In our ***initial evaluation*** we look for causes of ***higher inflammation*** and implement corrective steps.

One of the important triggers of inflammation with aging is diet. Some dietary patterns ***increase inflammation***, and others help to ***reduce inflammation***. With osteoporosis it is important to be on an anti-inflammatory diet. A measure of the inflammatory potential of the diet is the dietary inflammatory index (DII). This was derived by measuring inflammatory markers relative to different components in diet. Components that increased inflammatory markers result in a + score of up to total score of +8. Dietary components that decreased inflammatory markers get a – score up to a total of -8.

For example, a typical ***fast food diet*** has a DII of +5 or ***very inflammatory***. The components that contribute ***saturated fat*** (+3.7), ***high caloric energy*** (+1.8) and ***cholesterol*** (+1.1). In contrast, the ***Mediterranean diet*** had a DII score of -3.96 which is ***anti-inflammatory***. The components that had anti-inflammatory effect include ***fiber*** (-6.6) and ***phytonutrients such as flavonoids*** (-4.7).

To contrast some of the components of the two diets, the ***fast food diet*** contained ***32.74 grams of inflammatory saturated fat*** versus 20.84 grams or ***38% less in the Mediterranean diet***. Fiber (anti-inflammatory) in the Mediterranean diet was 26.81 grams versus 12.86 grams, or a whopping 117% more than in the fast food diet. The anti-inflammatory flavonoids were a striking 10-fold high in the Mediterranean diet.

As would be expected, the observational studies that look at bone mineral density in those consuming ***the Mediterranean diet*** have found that those following it have ***significantly higher bone mineral density*** in the spine and hip.(1)

The Mediterranean diet:

- Fruits and vegetables (especially non-starchy)
- Legumes (beans, lentils, chickpeas)
- Whole grains (bread, pasta, rice)

- Nuts and seeds
- Healthy Fats: extra virgin olive oil.
- Fish and seafood (especially oily fish like salmon and tuna)
- Poultry (in moderation)
- Dairy products (low-fat or full-fat, in moderation)
- Eggs (in moderation)
- Herbs and spices (e.g., oregano, thyme, rosemary)
- Moderate consumption of red wine (optional)
- Low intake of processed foods, sugary drinks, and refined grains

The added benefit of transitioning to an anti-inflammatory Mediterranean style diet is that it has been shown to be beneficial to all of the common diseases associated with aging.

Restoring More Normal Bone Remodeling

All of the drug therapies used in the treatment of osteoporosis target balancing ***one*** of the cell changes in bone remodeling:

1. ***Slowing*** osteoclastic ***bone removal*** (anti-resorptive therapy such as Fosamax)

2. ***Increasing*** osteoblastic ***bone building*** (anabolic therapy such as Evenity)

Ideally, therapies that ***target both pathways*** would be ideal. Several natural polyphenols have been ***shown to both*** quiet osteoclastic bone removal and stimulate osteoblastic bone repair. These include:

1. ***Curcumin***
2. ***Berberine***
3. ***Icariin from the herb Epimedium***
4. ***Fisetin from the herb Smoke Tree***

We use these phytonutrients as the core of our supplement program. We also use specific collagen peptides which have been shown to improve bone mineral density (BMD). During remodeling, osteoblasts must first build a collagen fiber mesh which the bone minerals are attached to. About 50% of bone strength is related to the quality of that collagen mesh.

Also important are the raw materials that are needed in the bone building portion of remodeling. They include:

1. ***Vitamin D***
2. ***Vitamin K***
3. ***Calcium***
4. ***Magnesium***
5. ***Other trace minerals such as silica***

Physical Modalities and Bone Building

Each remodeling cycle, new osteoblasts must be developed. They come from mesenchymal stem cells (MSCs) in bone marrow. With age this conversion from MSCs begins to diminish resulting in less osteoblastic bone replacement. The MSCs can be stimulated to help their conversion to osteoblasts by mechanical stimulation.

These cells have “mechanoreceptors”, cell surface receptors that increase MSC activity when they are mechanically stimulated. This is the basis of the ability of exercise to improve osteoporosis. While exercise is an essential part of osteoporosis treatment, the amount of BMD gain is insufficient to be a sole treatment, but it can contribute to a broader program and is an important element.

Exercise programs such as circuit weight training and daily walking with a light-weight vest have been shown to help. The specific exercise program should be adapted to the tolerance and ability of each individual.

Several therapy modalities which increase stimulation of cell mechanoreceptors have been researched in osteoporosis. The ***most effective*** is ***pulsed electromagnetic frequency or PEMF***. This is the core modality in our osteoporosis treatment program. PEMF has been used for some time to heal difficult fractures and to help increase the success of spinal fusion surgery. A PEMF device used to enhance bone healing in a cervical spine fusion is shown to the left.

More recently, equipment to apply it to broader areas including the mid, and lower spine and the hips which are the critical areas involved in osteoporosis has been developed. Multiple studies have ***found gains*** with a series of PEMF, showing BMD gains at ***15% or greater***. This ***higher gain is twice*** the typical gains seen with anti-resorptive medications which result in 5-7% gains. When ***PEMF is added to the nutrients*** discussed above and to regular weight bearing exercise, gains ***can result in 15-20% gains*** in bone mineral density.

These higher gains are important given how much bone is lost once the diagnosis of osteoporosis. The designation of ***osteoporosis*** is made when the ***T score on a DEXA scan is -2.5 or lower***. That ***represents a 20-25%*** loss of bone mineral density. First line drug treatments may produce 5-7% gains which improves but does not normalize a 20-25% loss.

The ***added benefit of PEMF*** is that these gains typically occur ***within the first year*** more quickly ***than are found with drug therapies***. This is important as ***maximum fracture risk reduction*** occurs when ***maximum therapy benefit*** is obtained.

Osteoporosis is a common disease associated with the aging process. ***Many patients forgo treatment*** concerned about the adverse effects of medication and the lack of symptoms in the earlier stages. There are ***3 potential paths*** anyone diagnosed with osteoporosis can take; begin drug therapy, begin a nutrition and PEMF program, and do nothing. Unfortunately, ***the do-nothing approach*** is ***commonly followed*** resulting in serious fractures. A recent study of 1100 patients over the age of 50 ***who sustained a hip fracture***, 70% had had a DEXA scan ***showing high risk of fracture*** yet ***only 10% had received any treatment***.(2)

Another complication with ***putting off early treatment*** is that the degree of treatment gain in bone mineral density ***declines with greater age***. In other words, a 75 year-old will not respond quite as much as a 60 year old.(3) It is always difficult receiving a diagnosis of a chronic disease, but it is important to begin working to manage it in the earlier stages. That philosophy is important in osteoporosis.

1) Noori et al. MEDITERRANEAN DIETARY PATTERN AND BONE MINERAL DENSITY: A SYSTEMATIC REVIEW AND DOSE-RESPONSE META-ANALYSIS OF OBSERVATIONAL STUDIES. Euro J Clin Nutri, 2022;76(12):1657-1664.
2) Arcidiacono et al. ESTIMATING THE EXTENT OF MISSED OPPORTUNITIES FOR OSTEOPOROSIS TREATMENT BEFORE HIP FRACTURE OCCURRENCE. Calcif Tissue Int. 2026 Aug 29;117(1):133.
3) Ishikawa et al. AGE-RELATED DIFFERENCES IN BMD RESPONSE DURING THREE YEARS OF DENOSUMAB TREATMENT. FRONT AGING. 2026 Aug 13;7:1892852.